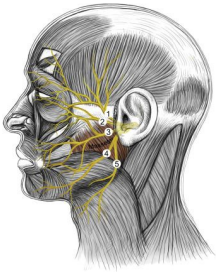
Facial Nerve



Objectives:

- Anatomy: course and branches
- Causes of facial palsy: including Bell's palsy, middle ear complication, traumatic and Ramsay Hunt syndrome

١٤٣٧أ: من ناحية الإختبار تم تسجيل الملاحظات المهمة آخر العرض واللي متأخر يقدر يذاكر من الملاحظات لان الدكتور حدد المهم ، والله يوفقكم

Resources: Doctor's slides, 436
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[Color index: Important | Notes | Extra]

Introduction

- The facial nerve (CN VII) provides motor fibers to the muscles of **facial expression**.
- It originates in the seventh nerve nucleus in the brain stem **(pons)**, enters the **middle ear** and mastoid and exits the skull at the **stylomastoid foramen** just in front of the mastoid process.
- From here it enters the **parotid gland** where it divides into its branches(Figure below).
- Paralysis can be caused by pathology anywhere along the nerve course or in the cortical nerves which control the nucleus (supranuclear or upper motor neuron fibers) resulting in **asymmetric movement** of some or all the muscles of facial expression.
- Facial nerve palsy causes difficulty with smiling, frowning and expressing emotions, it is a devastating condition for the patient.
- The causes are numerous and are listed in (Table 15.1). 'Supranuclear' or upper motor neuron causes will often spare the forehead as these muscles receive fibers from both facial nerve nuclei.

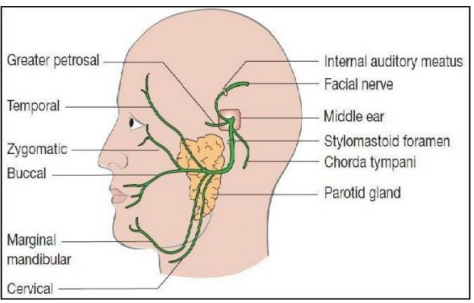


Table 15.1 Common causes of facial nerve paralysis

Supranuclear and nuclear (upper motor neurone)
- Vascular lesions, e.g. stroke
- Intracranial tumours
- Multiple sclerosis

Infranuclear (lower motor neurone)
- 'Bell's palsy'
- Trauma (birth injury, fractured temporal bone, surgical)
- Tumours (parotid tumours, acoustic neuroma, malignant disease of the middle ear)
- Middle ear suppuration (acute or chronic otitis media)
- 'Ramsay Hunt' syndrome
- Guillain–Barré syndrome
- Sarcoidosis

Facial Nerve (Anatomy)

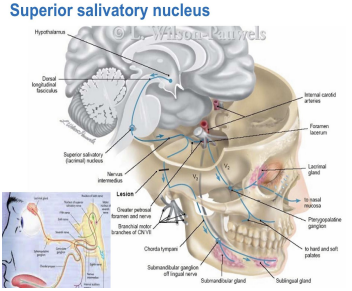
- Consists of 10k (10,000) neurons, 7k (7,000) of which have myelinated motor fibers (facial expression). Mixed nerve but mainly motor, 70% motor and go to the muscles of expressions, 30% sensory (10: Taste,10: General sensation,10: secretomotor)
- Superiorly along the roof of the IAC (7UP)
- The course of facial nerve: 7 segments (437A)

1.Pons
2.Cerebellopontine angle (CPA)
3.Internal Auditory Canal (IAC)
4.Labyrinthine fallopian canal
5.Tympanic fallopian canal
6.Mastoid fallopian canal
7.External

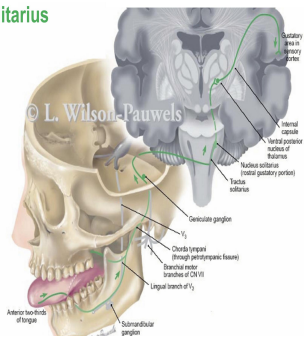
Nuclei (PONS) 4Ss' (The intracranial part)

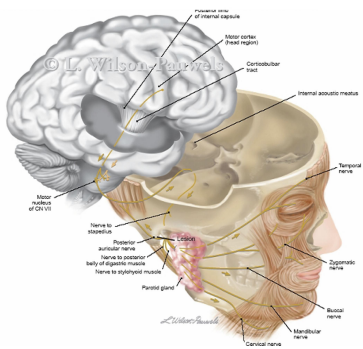
- This part includes the nuclei of facial nerve and the cerebellopontine (CP) angle segments.
- Facial Nerve Nuclei (in the pons): 4Ss
  - Solitarius (Taste) receives taste fibers.
  - Superior Salivatory (Lacrimal) Nucleus gives parasympathetic fibers responsible for lacrimation and salivation.
  - Spinal Nucleus of The Trigeminal Nerve. Send certain fibers to facial nerve. General sensation
  - Seventh (motor) 70% of facial fibers from this nucleus. facial nucleus: main nucleus which gives motor fibers
  - The fibers of solitarius, superior salivatory, and spinal nucleus form nerve of Wrisberg also called nervus intermedius then they join the fiber of the 7th nucleus and form 100% of the facial nerve. It is called facial nerve proper .
  - Imp: question can come from this part such as; The facial nerve that is responsible for the general sensation or special sensation comes from where?

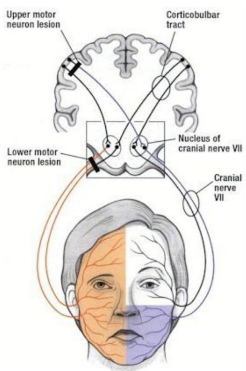
Superior salivatory nucleus



Solitarius







The Facial nucleus is divided into 2 parts: possible **MCQ**

1. The upper half that receives innervation from both cerebral cortices.
2. The lower half that receives innervation only from the contralateral cerebral cortex.

★ UMN means the lesion is above the nucleus.

★ LMN means the lesion is below or at the level of the nucleus, so any lesion from the internal auditory canal to the the parotid gland is considered lower

★ Lower motor lesions affect all the ipsilateral facial muscles "Lower motor neuron lesion is form the nucleus downward".

★ Upper motor lesions spare the upper facial muscles and affect the contralateral lower face because the forehead is innervated bilaterally.

- The forehead has bilateral innervation unlike the lower face which is supplied from the contralateral side.
- for ex a cerebrovascular accident causes UMN lesion.
- In LMNL the Idea is basically the involvement of frontalis (wrinkles of the forehead) and orbicularis oculi (give you the ability to close the eye), while in UMNL Frontalis and orbicularis occuli are spared.

- Examples:

Whole left side of the face is affected

Only the lower part of the left side is affected

Facial Nerve Fibers

Motor fibers:

- To the stapedius muscle of the middle ear and facial muscles.
- What does the stapedius do? a dampening action as it stabilizes the foot of stapes, so any damage to the stapedial nerve → hyperacusis and phonophobia

Secretomotor fibers (parasympathetic):

- To the lacrimal gland and the submandibular and sublingual salivary glands.
- Not the parotid gland, parotid gland is supplied by CN IX.
- Also supplies palatine and nasal glands.

Taste fibers:

- From the anterior two thirds of the tongue and palate, chorda tympani nerve. The posterior of the tongue innervated by glossypharyngeal.

Sensory fibers:

- General sensation: Pain, temperature, and touch from the external auditory canal (external auditory meatus).

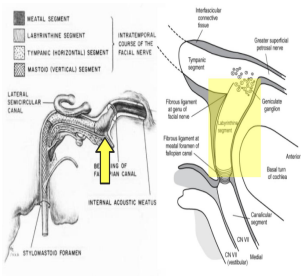
Motor part:

* Precentral gyrus (frontal lobe)
* Upper face corticobulbar cross
* Contralateral predominance
*Motor fiber bend around the abducens (CN VIth) nucleus

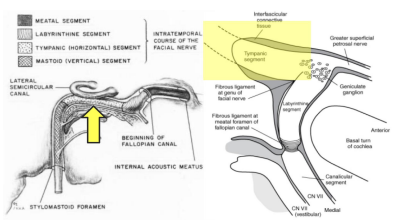
Facial nerve segments

Know the details they come MCQ

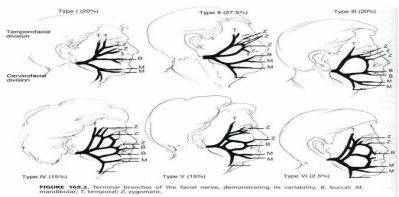
1. Intracranial (cisternal) segment.
2. Meatal segment (internal auditory canal): The first entrance to the IAC
   - Internal auditory canal (IAC) the facial nerve segment enters with the vestibulocochlear nerve
   - 7 UP: it is in the anterior superior portion of the IAC and behind it is the vestibulo (sup and inferior) and cochlear
   - Zero branches
   - 8mm
3. Labyrinthine segment:
   - Inner ear segment, shortest segment, 3-4mm
   - Starts from IAC to geniculate ganglion (one of the most important landmark).
   - Only segment that lacks arterial anastomosis, Imp because if there was edema and compression of nerve ischemia results.
   - 3 branches
   - Shortest segment and narrowest > susceptible to compression (only segment that lacks arterial anastomosis) > vulnerable to injury.
   - The geniculate ganglion before entering the middle ear gives the first branch (1st genu) > greater superficial petrosal nerve > fibers for lacrimation and salivation.
   - What is the first branch of CN VII? greater superficial petrosal nerve > lacrimal gland and salivation, any problem here would cause lacrimation deficit.
   - The patient can not lacrimate; eyes are dry then we would know the problem was before the greater superficial petrosal nerve.
   - If there is a lesion in the middle ear, after the first branch there will be a normal lacrimation and salivation, this process is called "Topography".

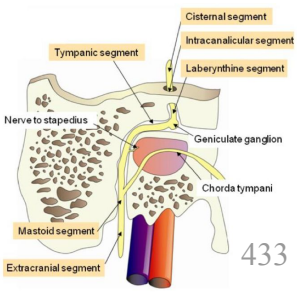


4. Tympanic segment:
   - Middle ear segment
   - Geniculate ganglion to pyramidal eminence (if you remove the drum you can see the facial nerve passing > importance; any infection or cholesteatoma can easily put pressure on the facial nerve)
   - zero branches
   - 50% dehiscent
   - 50% don't have a bone covering the nerve
   - (2nd genu)
   - 8-11mm



5. Mastoid segment:
   - Directed downwards vertically
   - 8-14mm, longest part of the intratemporal course
   - Pyramidal eminence to stylomastoid foramen, if a lesion is affecting the stapedius nerve or the chorda tympani it maybe high mastoid or tympanic
   - 3 branches:
     - Branche to Stapedius muscle: The stapedius muscle prevents high sounds from reaching the ear, injured patient can't tolerate high sounds
     - Branche to Chorda tympani nerve: 1.submaxillary and sublingual 2.taste anterior 2/3 tongue 3.pain, temperature, and touch EAC
     - Auricular nerve.
   - Patients with normal lacrimation but absent stapedial reflex would have a lesion here.
   - If patient has absent lacrimation, absent stapedial reflex, and absent anterior 2/3 of taste then the problem was before all the branching
6. Extratemporal (extracranial) segment:
   - 15- 20mm
   - 9 branches
   - Stylomastoid foramen to the parotid, which gets separated into superficial and deep parotid lobes, and it ends in 5 major branches (T,Z,B,M,C) imp to know them temporal, zygomatic, buccal, mandibular and cervical
   - Supply: Postauricular muscles, Stylohyoid muscle, Posterior belly of the digastric muscle.
   - Highest risk during surgical T&M
   - Pes anserinus (goose's foot): branching point of the extratemporal segment in the parotid, separates parotid into deep and superficial lobes.





The CP angle segments:

- Facial nerve is in relation with the last 4 cranial nerves.
- The facial fibers cross the cerebellopontine "CP" angle and pass through the internal auditory canal (meatal segment) with vestibulocochlear nerve (8th)
- 7 th CN occupies the anterior superior part of the internal auditory canal "7up" (possible **MCQ**)

Facial nerve segments

1. The intra**temporal** part:

From the internal auditory meatus or canal it crosses the temporal bone through fallopian canal and it is related directly to the inner, middle and external ear.

It is divided into 3 segments:

A. Labyrinthine: IAC to geniculate ganglion, in the inner ear, the only segment that lacks arterial anastomosis, (embolic phenomena, vascular Compression) high risk of ischemia (possible **MCQ**).

B. Tympanic: from geniculate ganglion to pyramidal eminence, in the middle ear, 50% of the Tympanic part is open in children, that's why they might get acute otitis media which can lead to facial nerve palsy. (imp for **OSCE**)

C. Mastoid or vertical: from pyramidal eminence to stylomastoid foramen, in the external ear it finally leaves the skull through stylomastoid foramen, mastoid has a high risk to get injured during surgery because it's the longest segment.

Branches are:

* Stapedius muscle: if get injured patient won't be able to tolerate high sound
* Chorda tympani nerve, which give:
    -Submaxillary,
    -sublingual and Taste anterior 2/3 tongue,
    - Pain, temperature, and touch EAC.
*Auricular nerve

MCQ:

- Labyrinthine segment is the shortest and narrowest part of the facial nerve segments, that's why most of the patients presenting with facial palsy due to compression of this segment.
- Whereas the mastoid segment is the longest → At risk of injury in trauma and mastoidectomy

2. The extra**temporal** (extracranial) part:

From stylomastoid foramen to division into major branches. As soon as the nerve leaves the stylomastoid foramen, it goes within the parotid gland and separates it into superficial and deep lobes. Then, it branches within the anterior border of the parotid into five terminal branches: (Always in Exam either MCQ or SAQ, know the nerve + the muscles + the functions)

a. **Temporal:** most superior > supplies the frontalis muscle.
b. **Zygomatic:** supplies orbicularis oculi muscle.
c. **Buccal:** supplies buccinators muscle. (if get injured food will accumulate on cheek + weak chewing) remember that the muscles of mastication are supplied by CNV
d. **Mandibular:** supplies the muscles of the angle of the mouth.
e. **Cervical** "long but thin branch": supplies platysma muscle.

- Most important 2 branches:
  1. Zygomatic "to protect the eye"
  2. Mandibular "its paralysis causes cosmetically bad deformity"
- Least important branch is cervical

There are usually some variations in different branches; some branches may get divided into two and each branch divides into another two etc.

Commonest surgical procedure that affects the mandibular branch → **submandibular salivary gland surgeries**, leading to **paralysis of the angle of the mouth**.

Parotid surgeries can cause facial nerve paralysis.

Embryology of facial nerve:
- Second Branchial Arch
- 0.05 % unilateral facial palsy
- 80% birth trauma
- 90% spontaneous recovery
- Congenital Unilateral Lower Lip Paralysis (CULLP)
- Anatomy of adult (Mastoid more superficial)
- The facial nerve is developmentally derived from the hyoid arch, which is the second branchial arch
- It arises as 2 main divisions-motor and sensory

*The motor division of facial nerve is derived from the basal plate of the embryonic pons.

*The sensory division originates from the cranial neural crest

The course of secretomotor and taste

- The secreto-motor fibers leave the superior salivary nucleus with the facial nerve. Some fibers leave the facial nerve in the geniculate ganglion as **greater superficial petrosal nerve** and this supplies the **lacrimal glands.** (if greater superficial petrosal nerve get injured patient will have Dryness) (imp of **MCQ**)
- The other fibers leave the facial nerve in the **chorda tympani** and supply the **submandibular and sublingual salivary glands**. Taste fibers follow the same course but in the other way. Taste fibers from anterior 2/3 of the tongue go through the chorda tympani to the facial nerve and finally to nucleus solitarius.
- What happens if there is an injury of the chorda tympani? It easily gets injured because it passes in the inner ear. Minor defect in the taste "because it affects the anterior 2/3 of only one side of the tongue" > There will be no dryness "because the parotid is supplied by the glossopharyngeal nerve (9th)"
- Function of Facial nerve:
  *Lacrimation
  *Expression
  *Mastication
  *Salivation
  *Speech
  *Hearing

Variations Anomalies

- **Dehiscence:** a defect in the fallopian canal, the nerve is exposed inside the middle ear, mainly congenital. When there is a deficiency of the bone, thus the nerve will not be covered by a bone and lies immediately in the middle ear.
- Fallopian canal is a bony canal through which the facial nerve passes inside the temporal bone
- Becomes more subjected to trauma and infection leading to facial nerve paralysis. (when they have otitis media they get facial paralysis)
- 50% of people are dehiscent

Facial Nerve Paralysis

Clinical manifestations:

1. Paralysis of facial muscles:
   a. Asymmetry of the face
   b. Inability to close the eye "orbicularis oculi"
   c. Accumulation of food in the cheeks "paralysis of buccinators, orbicularis oris"

- Lower motor neuron lesion of the left side: (upper picture)
  *No wrinkles in the forehead when looking up due to failure of contractions of frontalis muscle
  *Inability to close the eye completely "most accurate sign"
  *Flattening of the nasolabial fold
  *Angulation of the mouth when showing the teeth "the angle goes to the other side"
- Upper motor neuron lesion of left side: Looks normal at rest (lower picture 2)
  * The orbicularis oculi and frontalis muscles will not be affected.

2. Phonophobia:
   - Due to failure of stapedius attenuation reflex, uncomfortable feeling in exposure to loud sounds. Dumping effect loss
   - Acoustic reflex (stapedial reflex) is a useful tool to localize the lesion; if intact the problem is distal to it and vice versa.

3. Dryness of the eye:
   - Some people present with lacrimation and others present with dryness. Why?
   - Lacrimation is due to paralysis of **orbicularis oculi** as this muscle help in draining the tears.
   - Dryness is due to affection of **greater superficial petrosal nerve** which arise from geniculate ganglion.
   - So if the paralysis is above the level of geniculate ganglion > dryness
   - If below it > no dryness.

4. Loss of taste:
   - Very little because of chorda tympani branch of the facial nerve supplies just the anterior ⅔ of one side, they feel a metallic taste.

Evaluation

- Careful history - Physical exam - Audiometry - CT/MRI/other - Topographic - Electrophysiology
- Since the facial nerve is in close relation to the 8th nerve (vestibulocochlear). If a patient as facial paralysis the auditory function should be checked.
- Sometimes the lesion is originating from the 8th nerve leading to signs & symptoms.

Diagnostics

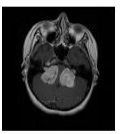
History & Clinical examination

History:

- Hearing loss or vertigo, SNHL, by audiometry & acoustic reflex
- Timing
  * sudden onset. aggressive? Or slow?
  * evolution over 2-3 weeks
- Presence of ear disease (3+5)
  * chronic\acute otitis media
  * cholesteatoma
- Vesicular eruption (2)
  * ramsay-Hunt syndrome
- Bilateral (4+6) diseases that causes bilateral nerve damage
  * Guillain-Barre syndrome
  * Lyme disease
  * Intracranial neoplasm
  * Neurofibromatosis
- Recurrence (1)
  * Melkersson-Rosenthal syndrome: 4Fs
    - Facial nerve palsy (Recurrent)
    - Furrowed tongue
    - Faciolabial edema
    - FHx + ve
- Severe pain, trauma, associated symptoms, past medical history

(2)

(1)



(4)

(3) Chronic otitis media

(6)

(5) Cholesteatoma

In History of Facial nerve palsy the most important question is WHEN DID IT START? Because if it starts from weeks and it's gradual you have to think of Malignancy

Examination: complete head and neck exam

- **Ask the patient to:**
  * Look up to test frontalis.
  * Close eyes to test orbicularis oculi.
  * Blow the cheek to test buccinators.
  * Whistling
  * Show the teeth for angulation (wide smile).
- **LMNL:**
  * Forehead wrinkling
  * Eye closure
  * Bell's phenomenon

House Brackmann Scale

House-Brackmann is the most useful classification in facial nerve palsy: (just know the name/ partial and complete/ that it is used because we can't just say nerve is paralyzed)

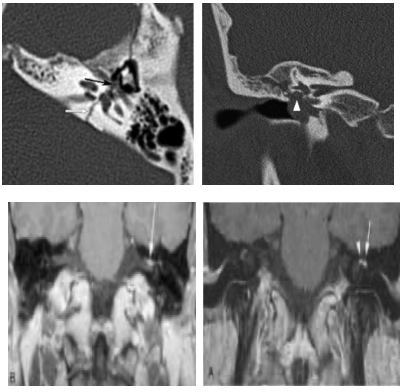
Grade 1: Normal.
Grade 2: Slight weakness.
Grade 3: Patient Can Close the Eye.
Grade 4: Patient Can't Close the Eye.
Grade 5 and 6: Masked Face, Asymmetric.

Grade	Appearance	Forehead	Eye	Mouth
I	normal	normal	normal	normal
II (Partial)	slight weakness normal resting tone	moderate to good movement	complete closure minimal effort	slight asymmetry
III (Partial)	non-disfiguring weakness normal resting tone	slight to moderate movement	complete closure maximal effort	slight weakness maximal effort
IV	disfiguring weakness normal resting tone	none	incomplete closure	asymmetric with maximal effort
V (Complete)	minimal movement asymmetric resting tone	none	incomplete closure	slight movement
VI (Complete)	asymmetric	none	none	none

Radiology

-Localize lesion

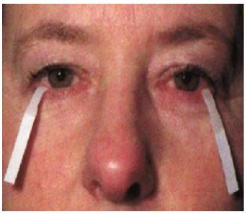
- Computed tomography (CT) (intratemporal)
  - Trauma
  - Mastoiditis
  - Cholesteatoma
- Magnetic resonance imaging (MRI) (intracranial)
  - Nerve enhancement due to infection
  - Exclude neoplasm (vestibular schwannoma or neurofibromatosis)



-Usually MRI enhancement in labyrinthine segment.

Topography

MCQs



- Not used as much now but it was used before to localize the lesion

1. Schirmer test → greater superficial petrosal (test lacrimation function)
   Put two tapes in the eyes and check if they're equal on both sides, if one eye is tearing and the other isn't; lesion proximal to this branch.
   Collect from both sides and compare the amount; If lacrimation is involved then the lesion is most likely proximal to the geniculate ganglion (before the greater superficial petrosal nerve branch)
2. Stapedial reflex → stapedial branch with tympanometry if normal we know problem after the branch
3. Electrogustometry "taste sensation" → chorda tympani
4. Salivary flow → chorda tympani
5. Imaging

Audiology

Evaluate for retrocochlear pathology (e.g. neoplasm) with either ABR or MRI. The facial nerve might be affected secondary to a lesion

- Evaluate for pathology of eighth cranial nerve
- Bell's palsy most common facial paralysis
  - Symmetric audiological function normal hearing
  - Absent ipsilateral acoustic (stapedial) reflex problem in labyrinthine segment or before stapedial branch.
- Retrocochlear pathology
  - Asymmetrical thresholds asymmetric hearing

Electrophysiology

- Provides prognostic information: not used for paresis only
- Principle: stimulate the nerve and look for response
- Tests:

  * Nerve Excitability Test (NET)
  * Electroneuronography (ENoG)
  * Electromyography (EMG) tests the strength of the muscle, important for prognosis
  * Maximum stimulation test (MST):

    ACUTE=Acute+Complete+Unilateral+Threedays+Evaluate
    used for prognostic follow up to see ow the nerve is healing

Nerve Excitability Test (NET)	Electroneurography (ENoG)
- Stimulate the nerve in the stylomastoid foramen and compare both sides. - The current's thresholds required to elicit just-visible muscle contraction on the normal side of the face are compared with those values required over corresponding sites on the side of the paralysis.	- The amplitude of action potentials in the muscles induced by the maximum current is compared with the normal side; and used to calculate the percentage of intact axons. - More objective

- It detects degeneration of the nerve fibers
- Useful only 48-72 hours following the onset of the paralysis.
- Provides prognostic information.
- If the nerve is stimulated distal to the injury in the first 2-3 days > there will be a response in all cases.
- After 3 days > there will be no response in case of degeneration.

Interpretation of the tests:

- Not useful in the first 48-27 hours.
- After 48-72 hours (the time required for degeneration to take place):
  *Normal results > no degeneration (neuropraxia)
  *Abnormal result > degeneration

Complications of Facial Paralysis

Facial paralysis severely affect:

- Normal facial expressions since the facial nerve supplies facial muscles of expression
- Mastication because patient can't close mouth
- Speech production
- Eye protection

Psychological trauma:

The most significant complication is the social isolation these patients

Pathophysiology of Nerve Injury

1. Neuropraxia (conductive block):

- In cases of mild trauma causing only functional block of the facial nerve, the fibers keep their integrity.
- In Regeneration: there will be restoration of full function if the cause is treated.

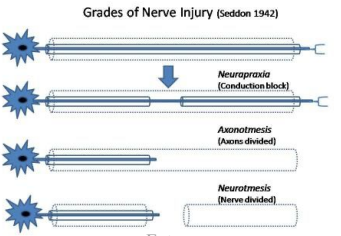
2. Neurotmesis (degeneration):

- Wallerian degeneration (distal to lesion), wallerian degeneration of the distal part of the nerve, takes 2-3 days to occur.
- Axon disrupted, loss of tubules, support cells destroyed
- Anatomical block due to complete transection of the facial nerve.
- In Regeneration: no recovery unless the distal and proximal parts are approximated.
- The recovery here is delayed and usually incomplete "why?" The rate of growing is 1mm/day or 1 inch/month > it will be delayed (It takes the fibers approximately 2-3 months to reach the angle of the mouth if the injury is in the temporal bone). Not all the fibers of the proximal part will reach the distal > it will be incomplete.
- After regeneration, sometimes misdirection of the fibers occurs: the fibers that go to the salivary gland deviate to sweat gland "the patient sweats when he eats" Or to lacrimal gland "patient tears when he eats (crocodile tears)"

3. Axonotmesis:

-Wallerian degeneration (distal to lesion)

-Endoneural sheaths intact



Extra

Facial nerve Palsy

Degeneration:

- Metabolic source (cell body).
- Wallerian degeneration:
  - o Begins within 24 hours
  - o Degeneration distal axon & myelin sheath
  - o Distal to the site of an injury.
  - o Without local Inflammation.
- Macrophages degrade myelin and axons

Regeneration:

- Axonal stumps swell and proliferating neuro-filaments
- Misdirected regrowth of nerve fibers
- Facial muscle contractures >> Synkinesia
- Salivation>>crocodile tears.

Principles of Management of facial nerve injury

- Take care of the eye, the patient is unable to close his eye so the cornea will be exposed to trauma, protect it by:
  1. Artificial tears if the patient has dryness.
  2. Protect them from dust by wearing sunglasses
  3. See ophthalmologist in case of any irritation
  4. Cover the eye while sleeping
- Treatment of the cause if applicable.
- Treatment of the nerve varies according to the degree of the paralysis.

-Partial facial paralysis:

-Being partial means that some of the nerve fibers are in continuity. Recovery is expected by conservative treatment (e.g. removal of pressure, steroid etc.). No need for surgical intervention.

-Complete facial paralysis:

-Complete paralysis may be a result of neuropraxia or/and degeneration.

-If it is **due to neuropraxia**, recovery is expected by conservative treatment, If it is **due to degeneration**, surgical treatment is required.

-To differentiate between degeneration and neuropraxia electrophysiological tests are required.

Causes of facial paralysis

According to the anatomy:

- Intracranial causes "brain tumors and neurosurgical trauma"
- Cranial (intratemporal) causes "middle ear infection or trauma"
- Extracranial causes "parotid tumors"

According to the cause itself:

- Congenital: Birth trauma.
- Traumatic: Forceps delivery, Basal skull/temporal bone fractures, Facial injury, Penetrating to middle ear, Barotrauma, Lightning, Head and neck injuries & surgery parotid, mastoid and intracranial surgeries.
- Iatrogenic
- Idiopathic
- Infection: Malignant otitis externa, Otitis media, Mastoiditis, Ramsey Hunt (Herpes zoster), Encephalitis, Polio, Syphilis
- Toxic
- Neurologic
- Neoplastic
- Inflammatory: O.M, Necrotizing O.E., Herpes.
- Neoplastic: Meningioma, malignancy of the ear or parotid.
- Neurological: Guillain-Barre syndrome, multiple sclerosis.
- Idiopathic: Bell's palsy "most common", Melkersson Rosenthal, Guillain Barre, MS,Mysethenia gravis, Sarcoidosis(Heerfordt's).
- Iatrogenic: Parotid surgery, Mastoid surgery, Local anesthesia, Acoustic neuroma

Sarcoidosis (Heerfordt's)

Bell's Palsy imp

- Most common diagnosis of acute facial paralysis, if slowly progressive it is NOT Bell's palsy.
- Diagnosis is by exclusion
- Unknown cause, LMNL, limited duration, minimal symptoms, spontaneous recovery, no sensory loss.
- **Pathophysiology:**

  Edema of the facial nerve sheath along its entire intratemporal course, "fallopian canal mainly labrithinate segment or inner ear" → Swelling of the nerve → Compression and ischemia → Complete paralysis, if mild edema → neuropraxia, if severe → degeneration.
- **Ethology:**

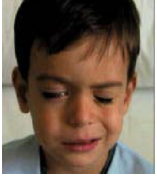
  Edema due to vascular (ischemia of the nerve) vs. viral measles in particular, cold weather but the exact etiology is still unknown (not proven)
- **Clinical feature:**
  - Can't close mouth and can't speak well
  - Sudden unilateral facial paralysis, occurs after exposure to cold weather could be vascular spasm, pain behind the ear → few hours later facial paralysis
  - Partial or complete
  - No other manifestations apart from occasional mild pain behind the ear. No discharge, no parotid swelling, not following trauma.
  - May recur in 10%(6 – 12%), previous history of paralysis in the same side 12%, other side 6%.
- **Risk factors:** family history and pregnancy
- **Diagnosis:**
  - Weakness of the entire half of the face
  - In doubt → CT and MRI scans
  - MRI may show contrast enhancement of the facial nerve
- **Treatment:**
  - Reassurance.
  - Eye protection. (Drops to prevent eye dryness and exposure keratitis)
  - Physiotherapy.
- **Medications:**
  - Steroids to decrease edema, antivirals, vasodilators. Antiviral and vasodilators only given in combination with steroids, not effective alone.
  - Corticosteroids (80 mg/day po), within 24 to 48 h of onset for 1 wk, decreased gradually over the 2nd wk
  - Antivirals (Acyclovir), less degrees of facial weakness

- **Surgical decompression in selected cases:** if patient is getting worse
  - Patients with 90% degeneration.
  - Within 14 days of onset.
  - Done by removing part of the bone around the nerve to decrease the compression
  - Surgery is not usually done because most of patients recover with conservative treatment
- **Prognosis:**
  - 80% complete recovery
  - 10% satisfactory recovery
  - 10% no recovery
  - Partial usually recovers within 4-6 weeks while complete may take up to 6 months
- Prevent corneal drying
  - Natural tears
  - Isotonic saline
  - Strips of skin tape to close the eye

Bell's palsy is a lower motor neuron facial palsy of unknown cause, but thought to be viral. (Will come in the exam). Bell's palsy may be complete or incomplete; the more severe the palsy, the worse the prognosis. In practice, full recovery may be expected in over 90% of cases. The remainder may develop persistent paralysis and other complications including ectropion (weakness of the muscles of the lower eyelid causing persistent overflow of tears) or an aberrant sequence of movements of the face (synkinesis). CT or MRI scanning may be needed if the symptoms persist or a specific cause (i.e. other than Bell's palsy) is suspected. Electrodiagnosis is used in the assessment of the degree of involvement of the nerve and includes nerve conduction tests and electromyography. These tests are done in a specialist center and be invaluable in predicting prognosis.

Inflammatory Causes of Facial Paralysis

1. Facial paralysis in Acute Otitis Media (AOM): **MCQ**
   - Mostly due to pressure on a dehiscent nerve by inflammatory products and fluid (pus) accumulation.
   - Usually is partial and sudden in onset.
   - Treatment: is by antibiotics and myringotomy; open the drum and drain the fluid.
   - Mastoiditis can cause pressure on the nerve.
   - 3 days history of fever, earache and facial palsy = AOM
   - left picture: pt has right facial paralysis and a red congested membrane.
   - In mastoiditis similar sxs to AOM but for treatment we put tube to release pus if fistula appears then mastoidectomy is indicated.

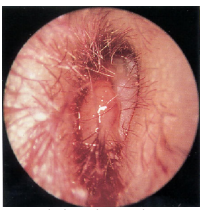



2. Facial paralysis in Chronic Suppurative Otitis Media (CSOM):

- Usually is due to pressure by cholesteatoma or granulation tissue causing bony erosion. tumor or mass
- Insidious in onset (slow) long history of deafness and discharge.
- May be partial "if detected early" or complete.
- Treatment is by immediate surgical exploration and "proceed"
- Mastoidectomy; remove the cholesteatoma and repair the nerve.

3. Malignant otitis externa (4Ds) "external auditory canal" also called osteomyelitis of temporal bone:

- Osteomyelitis of the temporal bone, osteomyelitis of the external auditory canal bone
- Granulation (at the junction of bony and cartilage) obscured TM, drum and tm can't be seen
- It could affect multiple nerves other than 7th if it reaches the jugular foramen (9th 10th 11th)
- Very aggressive, which is why it was thought to be malignant
- Scenario of old diabetic pt (immunocompromised) that has ear discharge and they show the above picture (we can't see the drum) say MOE or osteomyelitis of temporal bone


Granulation obscured TM
drum and tm can't be seen

- 4Ds:
  - Diabetes mellitus (DM) or anything considered immunocompromising
  - Discharge (purulent)
  - Discomfort
  - Dysfunction of cranial nerve

4. Herpes Zoster Oticus (Ramsay Hunt Syndrome):

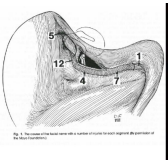
- Herpes zoster affection of cranial nerves VII, VIII, and cervical nerves, vestibulocochlear dysfunction (multi-nerves involvement).
- Symptoms: facial palsy, severe pain, skin rash (the hallmark), SNHL (sensorineural hearing loss) and vertigo (due to 8th nerve involvement), vertigo improves due to compensation from the other side "takes few weeks"
- SNHL is usually irreversible
- Facial nerve recovers in about 60%. Recovery of facial nerve function is much less likely than in Bell's palsy
- Treatment by: Acyclovir, steroid and symptomatic

- Similar symptoms to bell's palsy but it is more severe wit more symptoms, for example here there is a skin rash
- Treatment is equivalent Bell palsy acyclovir and steroids
- Poor outcome
- The patient is usually elderly, and severe pain precedes the facial palsy.
- The characteristic clinical feature is a vesicular eruption in the ear (sometimes on the tongue and palate).
- you will be asked about it in exam, patient has facial palsy and skin rash and Ear symptoms it's Ramsay hunt syndrome

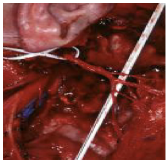
Traumatic Facial Injury 3 causes

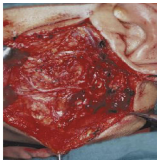
1. Iatrogenic:
   - Operations at the CP "cerebellopontine" angle, ear and the parotid glands.
   - Acoustic neuroma resection or mastoidectomy.
   - Local anesthesia.

mastoidectomy

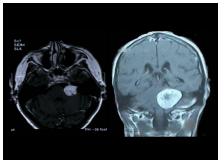


Parotid



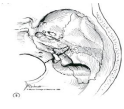


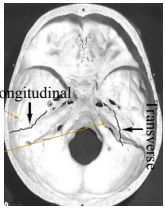
Acoustic neuroma (schwannoma)



2. Birth trauma (congenital facial palsy):
   - 80-90% are associated with birth trauma forceps use
   - 10 -20 % are associated with developmental lesions, ex neurofibromatosis
   - Most of them are partial and need only conservative management

3. Temporal bone fracture: 2 classifications **MCQ**
   - Longitudinal: يمشي مع العظم
     - 80% of Temporal Bone Fractures.
     - 15-20% Facial Nerve involvement.
   - Transverse: يقص العظم من النص
     - 20% of Temporal Bone Fractures.
     - 50% Facial Nerve Involvement (more likely to cause paralysis)
   - Most common cause of facial nerve palsy in temporal bone trauma is transverse temporal bone fracture, It is important to differentiate between the two.





- Signs for temporal bone fracture:
  - Raccoon eyes sign
  - Battle's sign
  - also called Skull base fracture signs
  - Ossicles injury
  - CSF or blood leak from ear

Pathology of traumatic facial nerve injury: Edema or transaction of the nerve

Management of traumatic facial nerve injury:

- If it is delayed in onset, it is usually incomplete and is due to edema → Conservative "steroids and relieve the pressure" directly after the trauma there is no problem because edema takes time
- If of immediate onset, it is usually complete and due to transection of the nerve:
  → Surgical repair
  → If borderline; conservative

SAQ

-What is the name of this sign?
-What does it indicate?
-What are the possible associated features?

1) hearing loss.
2) Facial nerve paralysis.
3) CSF leak.

Surgical Repair:

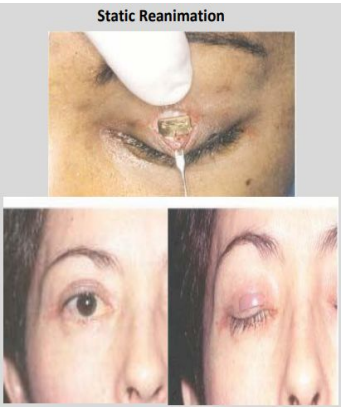
- **Direct Anastomosis:** If the proximal and distal parts are identified and no distance between them.
- **Nerve Graft:**
  - If there is a distance between them
  - Most common nerve used is great auricular nerve; it can give up to 10 cm and has the same thickness of facial nerve, or sural nerve.
  - If the injury is in the temporal bone, sometimes we graft the nerve in the fallopian canal without stitching
  - But if outside > we must stitch it.
- **Nerve Transfer (anastomosis):**
  - If the proximal part can't be identified, get a nerve and connect it to the distal part.
  - Most common nerve used is hypoglossal nerve 3, if the other one is healthy and functioning well, because bilateral hypoglossal nerve damage is catastrophic.
- **Muscle flap:**
  - If the distal and proximal parts can't be identified.
  - For cosmetics only "temporalis or masseter muscles are used".

Botulinum Toxin

Synkinesis and hypertonia

- Advantages
  - Ease of use
  - Selective
- Disadvantages
  - Temporary
  - Repeated every 3 months.

Synkinesis is a neurological symptom in which a voluntary muscle movement causes the simultaneous involuntary contraction of other muscles. An example might be smiling inducing an involuntary contraction of the eye muscles, causing a person to squint when smiling



Neoplastic

Malignant parotid lesion, Cholesteatoma, Acoustic neuroma, CN VII tumor, Meningioma.

SPORT > Neoplasm

- Slowly progressive
- Persistent >4 months
- Other C.N. Ex SNHL
- Recurrent
- Tumor History

Conclusion

- Facial paralysis sequelae (significant)
  - Functional
  - Cosmetic
  - Psychological
- The primary goals of facial reanimation
  - Corneal protection
  - Symmetry at rest
  - Smile restoration

Questions from the doctor's slides

A. What is the most likely diagnosis?

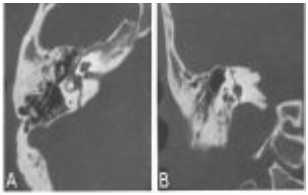

Left lower motor neuron facial paralysis (most likely bell's palsy).

Mention 2 common causes?

- All diseases of this lecture causes LMN facial paralysis
- Bell's palsy (most common)
- Temporal bone fracture
- Acute otitis media

B. 36 years old man with RTA:

What is your diagnosis?



Transverse fracture of the temporal bone.

Mention 2 clinical findings?

- LMN Facial nerve paralysis
- CSF leak
- Ossicles injury and other signs of skull base fracture (Racoon eye, battle's sign)

C. 34 years old with LMN facial paralysis:

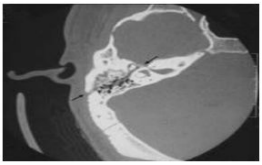
What is your diagnosis?

Herpes Zoster Oticus (Ramsay Hunt syndrome).

What is your management?

Acyclovir, Steroids, Physiotherapy.

D. 24 years old man involved in RTA: What is your diagnosis?



Longitudinal fracture of the temporal bone.

Mention 2 other clinical findings?

- LMN Facial nerve paralysis
- CSF leak
- Ossicles injury and other signs of skull base fracture

Doctor notes 437A (kindly read it)

1-What's the embryological origin of facial nerve?

Second branchial arch

2-Where is the position of the facial nerve during its course of the auditory canal?

The upper part (7UP)

Facial nerve segments:

3-What's the narrowest segment of the facial nerve, that is easily compressed?

Segment 4 labyrinthine

4-Which segment of the facial nerve is highly exposed and more prone to injury after otitis media?

Segment 5 tympanic

5-what's the largest segment of the facial nerve that is easily injured after a trauma?

Segment 6 mastoid

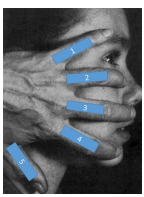
In question 3, name one of its branches that supplies the lacrimal gland and what will happen if it gets injured?

Greater superficial petrosal nerve, shedding of tears while eating (crocodile tears)

In question 5, name one of its branches that gives taste sensation and what will happen if it gets injured?

Chorda tympani, metallic taste

6-name the 5 major branches of facial nerve which are illustrated in this picture below?
might come as SAQ



1.Temporal
2.Zygomatic
3.Buccal
4.Mandibular
5.Cervical

7-what is the name of the classification used to grade facial nerve palsy?

Brackmann classification “in short”:

1= almost normal
2= partial degeneration
3≤ can close their eyes
4≥ can't close their eyes
6= almost all muscle affected (masked face)

8-whats the management of facial nerve palsy?

Eyes: artificial tears, might need a to put a special striprs to close the eyes while sleeping

Synkinesis: botox

9-whats the management of the patient had a complete nerve cut?

surgery (like surgical repair)

10-what's bell's phenomenon?

Upward and outward movement of the eye, when an attempt is made to close the eyes

11-what's the prognosis of facial nerve palsy in general?

90% completely recover

Might come as SAQ

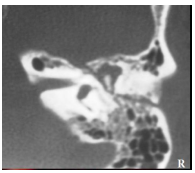
- 20 year old female brought to the ER after a car accident, her face was covered red because of the blood and it was clearly asymmetric, she lost one of the nasolabial fold, and she can't close her eyes at that side. An urgent CT was done, which showed the following (pic.)

What's the most likely diagnosis that explains the patient presentation?

Facial nerve palsy

After seeing the CT whats the most likely etiology?

Temporal bone fracture (longitudinal)



- Patient presented to the clinic complaining of ear pain vomiting and spinning sensation, on face inspection the doctor noted an abnormality (pic.)

What's the abnormality seen on this picture?

Asymmetric face (facial nerve palsy)

Which cranial nerves are most likely to be affected?

(CN 7-8) facial-vestibulocochlear

Which syndrome does this patient most likely have?

Ramsay hunt syndrome

- A patient presented with asymmetric face, metallic taste, drooling, hearing loss, an MRI was done which showed the following (pic.) what's the most likely lesion seen on this MRI?

Acoustic neuroma (schwannoma)